

Recently, at the radiological conference, Doctor Leaf of Stanford reported a five-year result in a patient with medulloblastoma, while Doctor Stone of the University of California, reported a similar case which had run two years. The latter patient was interesting, as he had very striking results after therapy; then he began to fail gradually, despite further treatment. Headache and blindness supervened. A second exploration showed no evidence of tumor, but a cyst—which, when evacuated, was followed by immediate improvement. To my way of thinking, the history of the last patient is very instructive, as it points out the fact that there must be mutual coöperation between the pediatrician, surgeon, and the radiotherapist, to assure the patient of a good end-result.

✱

HOWARD A. BROWN, M. D. (384 Post Street, San Francisco).—Signs of progressive increasing intracranial pressure, such as Doctor Scott has described, must be considered as suggestive signs of brain tumor. In some instances, however, it may be difficult to differentiate between increased intracranial pressure, resulting from brain tumor, and that seen in association with internal hydrocephalus or subdural hematoma. This is particularly true in infants where a gradual increase in the size of the head, enlarging fontanelles and separation of suture lines, with the development of "crack pot" resonance, may be the only signs of early pressure. Choked discs are frequently absent in infants, even in the presence of a high degree of pressure, because of the separation of the sutures which allows for expansion of the intracranial contents.

In cases of this type considerable information can be obtained by careful puncture through the lateral aspects of the anterior fontanel. A subdural hematoma can be located, if present, and the contents of the sac aspirated. If no hematoma is present, deeper puncture into the ventricle gives an estimate as to whether one is dealing with a hydrocephalus or not. If the ventricles are small, air can be introduced and ventriculographic studies made to localize the pathologic process. If a hydrocephalus is encountered, a small amount of a neutral dye may be injected into the ventricle and, subsequently, a lumbar puncture done to determine whether the hydrocephalus is of the communicating or obstructive type. Immediate recovery of the dye in the lumbar region indicates a freely communicative type of hydrocephalus, while failure of the dye to appear below indicates some obstruction to the outflow of cerebrospinal fluid from the ventricular system. The latter group may result from tumors of the posterior fossa or congenital malformations along the pathways of fluid escape from the ventricles and, in the majority of instances, may be relieved by surgical measures.

In our experience, brain abscess seldom gives rise to fever or leukocytosis, except in its early stages before it is well walled off from the surrounding brain. The presence of middle ear or mastoid infections or acute sinusitis preëxisting the signs of intracranial pathology will indicate the possibility of a brain abscess, although in rare instances they may occur without a definite infectious etiologic agent that can be recognized.

It is impossible to determine the type of tumor present until a surgical exposure has been made. The astrocytomas are fairly well circumscribed, and frequently may be removed completely without recurrence. This, of course, is in contrast to the medulloblastomas, which recur after surgical removal. In view of the marked difference in prognosis between the two types, operation is essential to offer a possible permanent cure in the astrocytomas and, at least, a relief of much of the headache and vomiting in the medulloblastomas.

Early recognition and surgical treatment of brain tumors offer a much better prognosis, both as to life as well as the preservation of vision and other special functions which may be permanently damaged by prolonged increased intracranial pressure.

Doctor Scott has called attention to the importance of a careful history and examination in cases presenting the symptoms of intracranial pressure which he has described. By such efforts, I am sure that our ability to diagnose and treat such conditions in the future will steadily increase in efficiency.

## RELAPSING FEVER IN CALIFORNIA\*

By G. CRESWELL BURNS, M.D.  
Walnut Park

DISCUSSION by Robert T. Legge, M.D., Berkeley;  
LeRoy Briggs, M.D., San Francisco; Howard O. Dennis,  
M.D., Beverly Hills.

RELAPSING fever occurs in many portions of the world. The existence of the disease has been known for centuries. Hippocrates described an epidemic of relapsing fever in Thasos. In 1741 Rutty observed a disease with relapses associated with typhus fever in Dublin. Livingston, in 1857, described a peculiar relapsing fever in South Africa, which he stated the natives considered to be carried by ticks. Then, in 1868, Obermeier observed a large spirillum in the blood of a patient, and an epidemic in Berlin in 1872 gave him the opportunity to confirm his observation in a number of patients. His findings were published in 1873.

In North America the disease was first recognized clinically in 1844 during an outbreak in Philadelphia, and was thought to have been brought into this country by Irish immigrants. In 1874 an epidemic of relapsing fever was observed among some Chinese laborers in Oroville, California.

Asiatic relapsing fever, which is louse-borne, is endemic, but at times becomes epidemic in European Russia, Poland, and the Balkan States. Sporadic cases and mild epidemics occur periodically in China, India, and north and west Africa. During the World War there was a severe epidemic in Serbia. In 1922 and 1923 there was an outbreak in the Gold Colony in west Africa.

Tick-borne relapsing fever, known as African relapsing fever, is present in southern, eastern, and a portion of central Africa. This variety of the disease occurs sporadically in Southern California and Mexico, and is endemic in Central and South America, Spain, Palestine, and Persia, but this type of the disease rarely becomes epidemic in character.

In September, 1922, Briggs<sup>1</sup> of San Francisco reported two cases of true relapsing fever, contracted by a man and his wife at Polaris, on the Truckee River. These are the first proved cases of relapsing fever arising from a focus in this State. Meader's<sup>2</sup> five cases in Colorado, which he reported in 1915, were the first proved instances of relapsing fever arising from an endemic source in the United States. Up until 1915 there had been a few scattered cases reported by the following observers:<sup>3</sup> Ward (1900), Tuttle (1906), Carlisle (1906), Goldfarb (1908), Hunter and Cope (1909), and Christian (1911). These cases, nine in number, occurring from 1900 to 1911, were all limited to direct importation—from Armenia, from eastern tropical America, from Turkey, from Russia, and from Macedonia.

Two years after Meader's report, Waring<sup>4</sup> reported another case in a boy who came from the same locality as Meader's patients. From 1917

\* Read before the Medical Section staff meeting of Los Angeles County General Hospital, January 28, 1935.

until 1922, when Briggs called attention to his cases, there was in the "Index Medicus" no reference to relapsing fever in the United States.

The first diagnosis of relapsing fever occurring in Panama was made in 1905. From that year until 1930 one hundred and seventeen cases were diagnosed at the Canal Zone Hospital.<sup>5</sup> The occurrence of the disease in Texas was reported by Kemp, Moursund, and Wright<sup>6</sup> in January, 1933. The first report of cases in Canada was made by Palmer and Crawford<sup>7</sup> in June of the same year.

In California no case of relapsing fever came to the attention of the State Board of Health from 1921 (the date of Briggs' cases) until September, 1930, when Dr. George Stevens<sup>8</sup> of the Los Angeles City Health Department reported a case in a school teacher who had lived at Big Bear Lake during July and August. The California State Department of Public Health up to November, 1932, however, had recorded thirty cases of relapsing fever. Varden's case<sup>9</sup> (reported in CALIFORNIA AND WESTERN MEDICINE, May, 1932), is included in this report. Legge<sup>10</sup> reported a case in May, 1933, and Coleman,<sup>11</sup> in December, 1933, reported the occurrence of the disease in himself, acquired accidentally through inoculation. These last two cases are also included in the California State Department of Public Health report of November, 1932.<sup>8</sup>

Up to January 1, 1935, a total of eighty-six cases of relapsing fever had been reported to the State Department of Public Health.<sup>12</sup> In the year 1933 there were nineteen, and in 1934 there were thirty cases.

California cases by geographic location according to counties are:

El Dorado and Placer .....	32
Mono .....	1
San Bernardino .....	46
Sierra .....	2
Tuolumne .....	1
Source out of the State (Nevada, 2; New Mexico, 1).....	3
Laboratory infection .....	1
Total.....	86

#### CLINICAL FEATURES

True relapsing fever is an acute infectious disease caused by a spirochete of the genus *Treponema*. The disease is characterized by paroxysms of fever lasting from three to six days, with an afebrile interval lasting about a week.

#### ETIOLOGY

*Borrelia recurrentis* is the name given to the organism causing European relapsing fever. Other strains have been thought to exist, and various strains have been studied in the different localities throughout the world where the disease has been isolated. Coleman,<sup>13</sup> in his experimental work on relapsing fever in California, concluded that the three California strains which he has in his possession are probably identical. He believes that the Novyi and Dutton spirochetes appear to belong

to different groups from the California spirochetes, though there may be some relation between the California and the Novyi strains. The tamarack gray squirrels and chipmunks are the animals which harbor the spirochetes in California, and the tick *Ornithodoros* is responsible for the transmission of the spirochetes to man. The mode of infection is contamination, the coxal fluid and feces of the tick being rubbed into the skin of humans. It seems that a bite is not necessary to effect transmission. The list of animals in various localities throughout the world which have been shown to be infested with the spirochetes of relapsing fever is large, including the armadillo, opossum, and dog. Curiously enough, the California ground squirrel is immune.

#### SYMPTOMS

The incubation period is usually less than twelve days; the average, seven. Prodromal symptoms are unusual, but in some cases the onset of the disease may be marked by mental depression, inactivity, slight shivering, and sweating. The onset of the attack is sudden with a chill or, as is usually the case, a chilliness lasting several hours. The fever mounts quickly and there is severe frontal headache with nausea and vomiting. Spells of shivering may be prolonged and vomiting often is severe. Pain in the epigastrium and in the region of the liver and spleen may be a complaint. Generalized muscular and joint pains may become excruciating. In young persons there may be convulsive seizures. The temperature becomes high, to 104 and 105 degrees Fahrenheit. The pulse is alarmingly rapid. Jaundice has been reported as a common symptom in some epidemics. Cough is often a symptom. The patient appears prostrated, the face is flushed and may be cyanotic. The temperature remains high for a few days—three to five or six, with irregular fluctuations. The skin is usually dry and hot. Nose-bleed is frequent, and there may occur hemorrhages into the skin, into the conjunctiva, from the ears and from the gastro-intestinal tract. The patient may become delirious. A macular eruption sometimes appears over the thorax, abdomen and legs, lasting a few days. The spleen is enlarged and tender; likewise the liver. Herpes is common. The urine is high-colored and contains albumen.

At the end of the paroxysm the temperature drops suddenly. With this come a profuse perspiration and a feeling of great relief to the patient. The appetite returns and the patient soon feels well again. There is an apparent recovery which lasts for approximately a week. Then a relapse follows with a sudden return of all the acute febrile symptoms. Occasionally there is recovery after one attack of fever. Untreated, the disease will usually run through a varying number of relapses. Instances of many relapses have been recorded. Osler<sup>14</sup> says that a second and third may occur, and that there are instances on record of a fourth and fifth. Whitmore,<sup>15</sup> writing in "Tice's System of Medicine," states that in about two-thirds of the cases one relapse ends the disease,

that a second relapse is not uncommon, that a third and fourth relapse are much less common, and that very rarely are there more than four relapses. St. John,<sup>16</sup> writing in Cecil's "Textbook of Medicine," says that a second relapse is uncommon, a third rare. In the California cases there were several cases with four relapses, a few with five, one with eight relapses, and one patient continued to have febrile attacks every ten days from August of one year until March of the following. Clinical records of only thirty of the eighty-six California cases were available for reference.

At times the symptoms during the relapses may be more severe than during the original attack; but as a rule they are less severe and of shorter duration.

#### DIAGNOSIS

The diagnosis is established by finding the causative organism in the blood during a paroxysm. The spirochetes may be found by examination of a fresh drop of blood or in stained smears. The ordinary Wright's stain will suffice. In mild attacks the spirochetes may be so scanty as to be readily missed.

#### DIFFERENTIAL DIAGNOSIS

Sporadic cases of relapsing fever are frequently confused with malaria. But in the former the afebrile interval between paroxysms is usually longer, and there is a leukocytosis, while in the latter there is generally a leukopenia. Dengue fever is differentiated by a leukopenia and an absence of spirochetes in the blood. Smallpox, influenza, and yellow fever have to be differentiated. The early stages of plague may be confused with relapsing fever. The enlarged spleen speaks against smallpox, influenza, yellow fever, and dengue. During epidemics typhus fever may cause confusion. Undulant fever needs to be differentiated. In children acute gastro-intestinal disturbances, pyelitis, early pneumonia, may be the first conditions to be considered. The polymorphonuclear leukocytosis is apt to be more confusing than it is a help.

#### COMPLICATIONS

In California the complications have apparently been slight if not nil. But in severe epidemics abroad serious complications have occurred. Bronchitis and pleurisy are fairly common. Pneumonia may develop. The spleen may rupture. There may occur cerebral hemorrhage or thrombosis. Heart failure and nephritis may follow prolonged relapses. Iridocyclitis is fairly common.

#### PROGNOSIS

Before the introduction of salvarsan treatment of relapsing fever, the mortality from the disease was from 2 to 5 per cent. In China and Indo-China the mortality often has been as high as 25 to 40 per cent. In the epidemics in India and China in 1912-1913, the mortality was as high as 70 to 80 per cent. Most of the deaths are due to complications. Children and adults in good physical condition do not often develop complications.

#### TREATMENT

Prophylaxis is of value in prevention of the disease, especially in conditions of filth and famine. In California, in the mountains, modern living conditions generally prevail in the cabins and cottages. The ticks are part of the great outdoor life, and their presence need not necessarily signify the existence of unhygienic conditions. Nevertheless, prophylaxis should be aimed directly at removing the spirochetes from man, as a source of infection, by curing the patients as soon as possible, and by protecting persons from the bites of bedbugs, lice, and ticks.

Specific treatment consists of giving arsphenamin or neoarsphenamin. St. John<sup>16</sup> states that in fully 80 per cent of cases the intravenous injection of 0.3 gram of salvarsan or 0.45 gram of neo-salvarsan will end the infection. It is advisable not to give too large doses, since relapsing fever lowers the resistance of the patient to the toxic properties of the drug. Best results are obtained if the drug is given early in the disease and at the onset of a paroxysm. For a child of seven, an injection of 0.15 gram of neoarsphenamin should ordinarily be well tolerated.

General measures are those employed in the treatment of other acute infectious diseases.

#### REPORT OF CASE\*

The patient is a boy, seven years old, who contracted relapsing fever at Big Bear Lake in San Bernardino County. He had four relapses, one of these occurring after specific treatment had been instituted.

The patient has had measles, chicken-pox, and whooping-cough. The latter disease, which occurred at the age of seventeen months, left him with certain allergic tendencies. Each upper respiratory infection would be accompanied by considerable asthmatic wheezing. In April, 1934, the patient was in a hospital because of bronchial pneumonia, verified by x-ray. The rest of the history is irrelevant.

The patient left home June 28, 1934, with three other children to spend the summer in a modern cottage at Big Bear Lake. On July 8, ten days after arriving in the mountains, quite by accident it was discovered that he had a temperature by mouth of 99 degrees. The next day the patient complained of malaise, headache, and was feverish. The local physician was called in to see the patient the following day because his temperature had gone to 103 degrees Fahrenheit (by mouth). The patient vomited several times, complained of severe headache and epigastric pain. He was brought home from the mountains July 10, and the following day continued to be nauseated and vomited once. His temperature had come down, and by July 12 he was feeling well again, appetite had returned, and he was ready to get out of bed. But he was kept quiet for another day. On July 14 he was out riding his bicycle, and apparently had recovered from the acute illness. It was felt that the condition was either an acute respiratory infection or an acute gastro-intestinal disturbance. Coming in the midst of an epidemic of poliomyelitis, this disease was also borne in mind.

On July 19 at three o'clock in the morning the patient awakened and complained of feeling hot. He was perspiring. He was given five grains of aspirin, and he fell asleep. At seven o'clock in the morning his temperature was 102 degrees Fahrenheit. That afternoon the temperature was 104.6 degrees. It dropped some during the night, and the next morning, July 20, at ten o'clock his fever was 105.2 degrees Fahrenheit.

\* Editor's Note.—Report of case is that of the author's own son.

The patient was prostrate, complained of severe headache and epigastric pain, with soreness in the upper left quadrant of the abdomen. He had a dry cough and his nose bled. Physical findings, as previously, were essentially negative, with the exception of tenderness on palpation in the region of the spleen, though no enlargement of that organ could be detected. A glass of hot lemonade with whisky seemed to be what induced a break in the fever, for in two hours his temperature was down to 100 degrees. The patient perspired profusely. As his temperature was rising during the morning hours, the patient had complained of feeling cold, but at no time during the initial attack or during any of the relapses had there been a definite chill. He felt well again by July 22 and was soon up and about, playing. He was taken back to Big Bear Lake on July 28 and had another recurrence of fever to 104.6 degrees the following day. His temperature the evening of July 29 was 96 degrees, recorded after several hours of profuse sweating. The patient was brought back home again the following day, July 30. His temperature rose again to 103.6, and a blood count done then was: hemoglobin, 80 per cent (T.); erythrocytes, 5,000,000; leukocytes, 14,400; with 75 per cent polymorphonuclears, 9 per cent mononuclears, and 16 per cent lymphocytes. The neutrophils were adult in form. Urinalysis was negative, with the exception of a faint trace of acetone.

Relapsing fever of some nature was obviously by now the clinical diagnosis. A sample of blood was taken for agglutination test. This was reported negative for typhoid, paratyphoid A and B, but was positive for mellitensis in strong dilution (1 to 20). The following day, July 31, three white mice were inoculated intraperitoneally at the bedside, each with one cubic centimeter of the patient's blood.

The patient again had an afebrile interval and felt quite well. Two of the mice became ill on the sixth day after inoculation, and were examined the afternoon of August 7. That evening the patient complained again of malaise, and the next morning, August 8, at eight o'clock his temperature was 102 degrees. Blood smears were made at the beginning of this relapse and were positive for spirochetes. The blood from two of the inoculated mice also was positive for the spirochetes of relapsing fever. A small dose of neoarsphenamin, 0.03 gram was given intravenously to the patient at ten o'clock, August 8. This dosage was decided upon after reference to the article by Legge, in which he stated that this amount of the drug had been effective for his patient, who was an adult. Later it was concluded that in Legge's article there had been a typographical error and that what his patient actually received was 0.3 gram and not 0.03 gram of neoarsphenamin. The afternoon of the day the patient was given this small dose of neoarsphenamin his temperature reached 103.4 degrees. The next day his temperature climbed to 105.4 degrees, with all the previous symptoms and findings present, but on August 10, the following day, his temperature did not go above 98 degrees. It had been planned to wait four days before repeating the neoarsphenamin, but on the third day following the initial injection another unexpected relapse took place, patient's temperature mounting to 105.2 during the night. He again felt quite well the next day, though by now he was looking quite pale and worn out as the result of the repeated febrile attacks. On August 13 he was given 0.15 gram of neoarsphenamin intravenously, and this dose was repeated in four days. On the day his fifth relapse would have occurred, his temperature rose to 99 degrees. On August 21 he was given another intravenous injection of neoarsphenamin, the dose this time being 0.1 gram. He was allowed to return to Big Bear Lake the following day, where he remained symptom-free and quickly regained his lost weight and strength.

#### COMMENT

The small initial dosage of neoarsphenamin produced a reaction similar to a "provocative." Until this intravenous injection was given, the relapses

were occurring at regular ten-day intervals, but this injection apparently was responsible for precipitating the unexpected relapse which followed two days after the peak of the last febrile attack. Perhaps the second dose of 0.15 gram would have sufficed to prevent further relapses, but to make certain that the blood stream was rid of the spirochetes a third injection was felt advisable. The child received a total of 0.45 gram.

None of the other children or adults in the cottage contracted the disease, but a five-year-old girl, living for the summer in a cottage about one hundred yards away, also developed the disease. The diagnosis was made by Doctor Varden of San Bernardino. It was learned that a girl of three, staying for the summer in a cottage just next door to the cottage where the patient was living, had contracted relapsing fever three years previously.

Miss Dorothy Beck of the California State Board of Public Health conducted a search of the cottage for ticks and other insects. Some ticks were found in the vicinity of the cottage, but whether they were *Ornithodoros moubata* or some other species I am not able to state. Miss Beck informs me that the ticks have not been classified as yet.

I have learned from Miss Beck that practically all of the San Bernardino County cases have come from the vicinity of Big Bear Lake.

In conclusion, I should like to say that the question of latent infection is apt to be of some concern, but in this regard Coleman<sup>11</sup> has concluded, from his experimental work on mice, that the brain was never virulent unless the blood was. He attempted to reactivate spirochetes in mice which had apparently recovered recently or after a long interval, and no reinfection was revealed.

7878 Seville Avenue.

#### REFERENCES

1. Briggs, LeRoy H.: Relapsing Fever in California, *J. A. M. A.*, 79:941-944, (Sept. 16), 1922.
2. Meader, C. H.: Relapsing Fever in Colorado, *Colorado Med.*, 12:365, 1915.
3. Quoted from Briggs' paper.
4. Waring, J. J.: *Am. J. Med. Sc.*, 155:819, 1918.
5. Dunn, Lawrence H., and Clark, Herbert C.: Relapsing Fever in Panama, with Special Reference to Animal Hosts, *Amer. J. Trop. Med.*, 13:2 (March), 1933.
6. Kemp, Hardy A., Moursund, W. H., and Wright, Harry E.: Relapsing Fever in Texas—I. The Identity of the Spirochete, *Amer. J. Trop. Med.*, 13:4 (July), 1933.
7. Palmer, J. H., and Crawford, D. J. M.: Relapsing Fever in North America, with Report of an Outbreak in British Columbia, *Can. Med. Assoc. J.*, 28:6 (June), 1933.
8. Porter, Giles S., Beck, M. Dorothy, and Stevens, I. M.: Relapsing Fever in California, *Amer. J. Pub. Health*, 22:1136 (Nov.), 1932.
9. Varden, Arthur E.: Relapsing Fever, *Calif. and West. Med.*, 36:5 (May), 1932.
10. Legge, Robert T.: Relapsing Fever, *Calif. and West. Med.*, Vol. 38, No. 5, pages 380 and 370 (May), 1933.
11. Coleman, George E.: Relapsing Fever in California—I. The Experimental Disease, *J. Infect. Dis.*, 53:337 (Nov. and Dec.), 1933.

12. Dunshee, J. D., Director of Public Health, State of California: Personal communication.
13. Coleman, George E.: Relapsing Fever in California—II. Immunity, *J. Infect. Dis.*, 54:1 (Jan. and Feb.), 1934.
14. Osler, Sir William, and McCrae, Thomas: *The Principles and Practice of Medicine*. D. Appleton & Co. Ninth edition.
15. Tice's System of Medicine. W. F. Prior & Co., Inc.
16. Cecil, Russell L.: *Textbook of Medicine*. W. B. Saunders Co. Second edition.

## DISCUSSION

ROBERT T. LEGGE, M.D. (University of California, Berkeley).—Our State has had several diseases that are essentially Californian, *e. g.*, tularemia, coccidiosis, and relapsing fever. It was due to members of this society and investigators within our State that these diseases were either definitely determined as entities or their relative causes discovered.

The medical and public health professions at the moment are much concerned as to the number of cases of relapsing fever that are being reported. Investigations have shown that this disease is endemic in California.

Physicians, therefore, must be on the alert and suspicious of certain fever patients, especially those living within endemic areas, or who have visited there. The diagnoses can be determined positively by clinical laboratory examinations of blood smears taken when the patient has temperature, for the presence of spirochetes, and by mouse inoculation as pointed out by Doctor Burns, as these animals are highly susceptible to the disease.

In my case, reported in this JOURNAL, May, 1933, the patient, Mr. C. M. Wheeler, M.S., a graduate student with Doctor Herms, doing research in medical parasitology at the University of California, who was accidentally infected while engaged in field work, is now a candidate for a Ph.D. degree. His thesis is to be on experimental evidence as to the vector that causes relapsing fever. This scientific piece of research will throw much light on an important subject.

The habitat of this rodent parasite is found usually in high altitudes, a significant fact, as most of the cases reported came from such endemic areas as Big Bear Lake and other Sierra regions. Herms, Meyer, and Wheeler have positively recovered this parasite in chipmunks' nests, and have incriminated the vector now known as *Ornithodoros hermsi*, so named after Professor William B. Herms of the University of California.

✱

LEROY BRIGGS, M.D. (384 Post Street, San Francisco). Relapsing fever yearly is becoming more of a vacation health problem in an ever-increasing area throughout the West, and continued reference to it by local authors is necessary to make us diagnostically conscious. Not that the disease is a serious one, since the mortality so far has been nil; but early recognition and treatment is essential to cut down the period of disability. In regard to the physical findings as outlined by the author, experience has shown that in the mild variety we have here in California, eruptions are rare—herpes occur only in about 14 per cent, and splenomegaly in 33 per cent.

In the transmission of the disease a proved vector has at last been found<sup>1</sup> as a new species of *Ornithodoros* tick. Specimens of this were collected from the two larger endemic centers, and monkeys and mice successfully inoculated by allowing the ticks to feed upon them. In honor of Professor Herms, the name "*Ornithodoros hermsi*" has been proposed for this species. It must be repeatedly emphasized that in the minds of the laity the word "tick" is ineradicably associated with the "wood tick" of the genus *Dermacentor*, whose habits of feeding are so different from the *Ornithodoros*. The latter drops off after feeding and, with the exception of *O. coriaceus* or the

pajaroello, leaves no particular lesion at the site of the bite, while the *Dermacentor* is found embedded in the skin. Furthermore, the larval and nymphal stages are capable of transmitting the disease, yet are so small as to be nearly invisible. These facts must be considered in the proper evaluation of the usual history of "having seen no ticks or bites."

The chief hope for prophylaxis lies in an attack on the rodent reservoir. The tick vector is so rarely discoverable we do not know whether man to man infection via vector plays a part; but we do know that certain rodents, such as chipmunks and tamarack squirrels, are widely and constantly infected. These can be destroyed, and steps are now being planned for control measures around the major endemic centers through the joint action of the Federal Government and the State Board of Health. It is to be hoped that the sentimentality of certain "nature lovers" will not be allowed to interfere with this step forward.

✱

HOWARD O. DENNIS, M.D. (Beverly Professional Building, Beverly Hills).—Doctor Burns' paper again emphasizes the fact that relapsing fever has become a diagnostic problem in California. This is true particularly in the summer months, when vacationists travel to the higher altitudes, where reservoirs of the infection have been found to exist.

In Southern California, Big Bear Valley has been the source of practically all of the cases. Only one case has been reported from Lake Arrowhead. It is important, therefore, to stress the value of a history of having visited in such areas approximately a week before the onset of the symptoms. Such a history in cases exhibiting signs of an acute infection as that which Doctor Burns has described, should at least raise the question of relapsing fever as a possibility.

My personal experience has been limited to one case from this area. This patient demonstrated the difficulty in finding the organisms in some cases despite careful study of a large number of smears made at the onset of each of several paroxysms. The ultimate diagnosis was made on the finding of but two or three spirochetes in one of several slides. Unfortunately, mouse inoculation was not done as early as would be the case should similar circumstances again develop. In view of this difficulty, mouse inoculation is to be resorted to as soon as a suspicion of this condition has been aroused.

In Doctor Burns' case report he mentions the positive agglutination of mellitensis in a dilution of 1-20. As undulant fever is considered in a differential diagnosis, such a finding might indicate that one were dealing with an infection due to this organism. It should be pointed out that a diagnosis of undulant fever should not be made unless a positive reaction in a dilution of approximately 1-1000 is found. Positive reactions of 1-20 and slightly higher are frequently found in individuals for a period of several years after the original infection with mellitensis has occurred. A recent check on Doctor Burns' child shows the reaction now to be negative.

California cases reported to date during 1935 are as follows: El Dorado County, two; Nevada County, one; San Bernardino County, four; Tuolumne County, one; State of California, one.

The patient from the State of California had been traveling through the Sierra Nevada mountain area and into Nevada; therefore, exact source of infection could not be determined, and the case was charged to California as a whole.

In addition, the names of seven other patients with diagnosis confirmed by laboratory findings have been reported, but sufficient data have not been received to determine localities in which infections were contracted. These seven were not included in the above tabulation; hence, the total for the year to date is sixteen cases. There have been no deaths.

Certainly, it has been shown by Doctor Burns' paper, and the paper of Dr. LeRoy Briggs earlier in the year, that the incidence is great enough to impose upon practicing physicians a definite responsibility for the diagnosis of relapsing fever which cannot be underestimated.

<sup>1</sup> Wheeler, C. M., Herms, W. B., and Meyer, K. F.: A New Tick Vector of Relapsing Fever in California, *Proc. Soc. Exper. Biol. and Med.*, 32:1290-1292, 1935.